

NEUROPEPTIDE GALANIN: POSSIBLE ROLE IN CONTROL OF FOOD INTAKE BY FATTY ACID OXIDATION. Sue Ritter. Department of V.C.A.P.P., Washington State University, Pullman, WA 99164-6520.

Recent studies support the existence of at least two distinct metabolic controls of food intake. One is activated by 2-mercaptoacetate (MA), a drug which blocks fatty acid oxidation. The other is activated by 2-deoxy-D-glucose (2DG), a drug which blocks glucose utilization. Our studies have shown that feeding and brain proto-oncogene expression induced by MA, but not by 2DG, is dependent on capsaicin-sensitive vagal sensory neurons and higher order neurons in the lateral parabrachial nucleus and central nucleus of the amygdala. Furthermore, we have shown that MA-induced feeding is blocked by co-infusion of either glucose or lipids. In contrast, 2DG-induced feeding is attenuated by glucose, but not by lipid infusion. Finally our results indicate that MA and 2DG activate distinct macronutrient appetites, that their potencies are differentially altered by dietary fat, and that they exert very different effects on plasma catecholamines and circulating metabolic fuels. Most recently we have found that MA, but not 2DG or cholecystokinin, greatly increased the number of nodose ganglion cells bodies expressing galanin mRNA. These results suggest that galaninergic vagal sensory neurons may mediate physiological responses to MA, perhaps including MA-induced feeding. Tempel and Leibowitz (1990) have shown that hypothalamic injections of galanin selectively increase fat intake and Tucker et al. (1992) have noted a significant positive correlation between hypothalamic galanin mRNA expression and daily fat ingestion. Thus galanin may participate at several levels of the nervous system in mediating responses to dietary fat or fat metabolism.